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=> s (human or sapiens) (4A) (serine kinase)

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=> s (human or sapiens) (5A) (serine (3A) kinase)

11 FILES SEARCHED...

24 FILES SEARCHED...

36 FILES SEARCHED...

49 FILES SEARCHED...

59 FILES SEARCHED...

81 FILES SEARCHED...

L1 6455 (HUMAN OR SAPIENS) (5A) (SERINE (3A) KINASE)

=> s (rick or rip2) (4A) (splice or splicing or spliced or variant)

27 FILES SEARCHED...

59 FILES SEARCHED...

L2 13 (RICK OR RIP2) (4A) (SPLICE OR SPLICING OR SPLICED OR VARIANT)

=> s l1 and l2

56 FILES SEARCHED...

L3 0 L1 AND L2

=> s (HUMAN OR SAPIENS) and l3

18 FILES SEARCHED...

24 FILES SEARCHED...

43 FILES SEARCHED...

59 FILES SEARCHED...

L4 0 (HUMAN OR SAPIENS) AND L3

=> duplicate

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PROCESSING COMPLETED FOR L2

L5 5 DUPLICATE REMOVE L2 (8 DUPLICATES REMOVED)

=> s l5 NOT (complete genome)

14 FILES SEARCHED...

33 FILES SEARCHED...

55 FILES SEARCHED...

78 FILES SEARCHED...

L6 5 L5 NOT (COMPLETE GENOME)

=> d l6 1-5 bib ab

L6 ANSWER 1 OF 5 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

AN 2000:42133 BIOSIS

DN PREV200000042133

TI RIP2 is a Raf1-activated mitogen-activated protein kinase kinase.

AU Navas, Tony A.; Baldwin, Daryl T.; Stewart, Timothy A. [Reprint author]

CS Dept. of Endocrine Research, Genentech, Inc., 1 DNA Way, South San Francisco, CA, 94080, USA

SO Journal of Biological Chemistry, (Nov. 19, 1999) Vol. 274, No. 47, pp. 33684-33690. print.

CODEN: JBCHA3. ISSN: 0021-9258.

DT Article

LA English
ED Entered STN: 26 Jan 2000
Last Updated on STN: 31 Dec 2001
AB RIP2 is a serine-threonine kinase associated with the tumor necrosis factor (TNF) receptor complex and is implicated in the activation of NF-kappaB and cell death in mammalian cells. However, the function of its kinase domain is still enigmatic as it is not required in engaging these responses. Here we show that RIP2 activates the extracellular signal-regulated kinase (ERK) pathway and that the kinase activity of RIP2 appears to be important in this process. RIP2 activates AP-1 and serum response element regulated expression by inducing the activation of the Elk1 transcription factor. RIP2 directly phosphorylates and activates ERK2 in vivo and in vitro. RIP2 in turn is activated through its interaction with Ras-activated Raf1. Kinase-defective point and deletion **variants** of RIP2 also significantly blocked the activation of ERK2 by TNFalpha but not epidermal growth factor. These results describe a novel pathway of ERK activation and the first catalytic function ascribed to any of the RIP-like kinases associated with the TNF receptor superfamily.

L6 ANSWER 2 OF 5 PROMT COPYRIGHT 2004 Gale Group on STN

AN 2001:397219 PROMT
TI NEXPO 2001: Profiles.
AU Santo, Jamie
SO Editor & Publisher, (14 May 2001) pp. 24.
ISSN: 0013-094X.
PB BPI Communications, Inc.
DT Newsletter
LA English
WC 16934
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AB Booth: 1600
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L6 ANSWER 3 OF 5 PROMT COPYRIGHT 2004 Gale Group on STN

AN 2000:57338 PROMT
TI Manufacturers and Suppliers. (Alphabetical list of companies)
SO Lasers & Optonics, (Nov 1999) Vol. 18, No. 11, pp. S8.
ISSN: 0892-9947.
PB Cahners Publishing Company
DT Newsletter
LA English
WC 71777
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AB A
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Subscription: \$61.00 per year. Published monthly.

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AN 2003:238035 USPATFULL
TI Inhibition of interleukin-1-beta secretion by card proteins
IN Alnemri, Emad S., Ambler, PA, UNITED STATES
PI US 2003166192 A1 20030904
AI US 2002-50054 A1 20020116 (10)
PRAI US 2001-340161P 20011214 (60)
US 2001-262477P 20010116 (60)
DT Utility
FS APPLICATION
LREP SEED INTELLECTUAL PROPERTY LAW GROUP PLLC, 701 FIFTH AVE, SUITE 6300,

SEATTLE, WA, 98104-7092
CLMN Number of Claims: 112
ECL Exemplary Claim: 1
DRWN 7 Drawing Page(s)
LN.CNT 3347

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides isolated Pseudo-ICE and ICE-Like, functional fragments thereof, or immunogenic fragments thereof and nucleic acid molecules encoding the above polypeptides. Also provided are various methods of using these polypeptides or nucleic acid molecules in modulating apoptosis or inflammation.

L6 ANSWER 5 OF 5 WPINDEX COPYRIGHT 2004 THOMSON DERWENT on STN

AN 1995-291207 [38] WPINDEX

DNN N1995-220081

TI Livestock rearing building, especially for pigs - uses stalls made from telescopic barriers erected along base of rick.

DC P14

IN STASHEVSKII, I I

PA (STAS-I) STASHEVSKII I I

CYC 1

PI RU 2028775 C1 19950220 (199538)* 8p

ADT RU 2028775 C1 SU 1992-5059636 19920620

PRAI SU 1992-5059636 19920620

AB RU 2028775 C UPAB: 19950927

The building has stalls for the animals, set in a row along the base of a hay or straw rick, each stall being formed by a barrier made from uprights (3) and transverse and lengthwise mesh walls with horizontal bars, doors and couplings. The stalls are equipped with mangers and between the rows of stalls there are gangways for feed distribution.

The supports (3) are telescopic and have upper sections which are moved by means of screws and gear nuts which are linked to a drive mechanism (37). The lengthwise walls of the stalls are also telescopic and equipped with drives (9).

The building can have a second row of stalls for the animals, with both rows having access ports to the rick, and, in a **variant** it can be made oval or round in shape and equipped with a central store for the feed.

ADVANTAGE - More convenient year-round rearing, with easy access to feed and bedding. Bul. 5/20.2.95
Dwg.2/13

=>

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LN.CNT 3073

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention is directed to novel polypeptides having homology to members of the tumor necrosis factor receptor family and to nucleic acid molecules encoding those polypeptides. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide molecules comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention.

L9 ANSWER 11 OF 12 USPATFULL on STN

AN 2002:235484 USPATFULL

TI Nod2 nucleic acids and proteins

IN Nunez, Gabriel, Ann Arbor, MI, UNITED STATES

Inohara, Naohiro, Ann Arbor, MI, UNITED STATES

Ogura, Yasunori, Ann Arbor, MI, UNITED STATES

PI US 2002127673 A1 20020912

AI US 2001-14269 A1 20011026 (10)

PRAI US 2000-244289P 20001030 (60)

DT Utility

FS APPLICATION

LREP David A. Casimir, MEDLEN & CARROLL, LLP, Suite 350, 101 Howard Street, San Francisco, CA, 94105

CLMN Number of Claims: 26

ECL Exemplary Claim: 1

DRWN 21 Drawing Page(s)

LN.CNT 5519

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to intracellular signalling molecules, in particular the Nod2 protein and nucleic acids encoding the Nod2 protein. The present invention provides isolated nucleotide sequence encoding Nod2, isolated Nod2 peptides, antibodies that specifically bind Nod2, methods for the detection of Nod2, and methods for screening compounds for the ability to alter Nod2 associated signal transduction.

L9 ANSWER 12 OF 12 USPATFULL on STN

AN 2002:75557 USPATFULL

TI Molecules of the card-related protein family and uses thereof

IN Bertin, John, Watertown, MA, United States

PA Millennium Pharmaceuticals, Inc., Cambridge, MA, United States (U.S. corporation)

PI US 6369196 B1 20020409

AI US 1999-245281 19990205 (9)

RLI Continuation-in-part of Ser. No. US 1998-207359, filed on 8 Dec 1998

Continuation-in-part of Ser. No. US 1998-99041, filed on 17 Jun 1998

Continuation-in-part of Ser. No. US 1998-19942, filed on 6 Feb 1998, now patented, Pat. No. US 6033855

DT Utility

FS GRANTED

EXNAM Primary Examiner: McGarry, Sean

LREP Fish & Richardson P.C.

CLMN Number of Claims: 14

ECL Exemplary Claim: 1

DRWN 18 Drawing Figure(s); 35 Drawing Page(s)

LN.CNT 6038

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Novel CARD-3, CARD-4L, CARD-4S, CARD-4Y, CARD-4Z, and murine CARD-4L polypeptides, proteins, and nucleic acid molecules are disclosed. In addition to isolated CARD-3, CARD-4L, CARD-4S, CARD-4Y, CARD-4Z, and murine CARD-4L proteins, and the invention further provides CARD-3, CARD-4L, CARD-4S, CARD-4Y, CARD-4Z, and murine CARD-4L fusion proteins, antigenic peptides and anti-CARD-3, anti-CARD-4L and anti-CARD-4S, anti-CARD-4Y, anti-CARD-4Z, and anti-murine CARD-4L antibodies. The

invention also provides CARD-3, CARD-4L, CARD-4S, CARD-4Y, CARD-4Z, and murine CARD-4L nucleic acid molecules, recombinant expression vectors containing a nucleic acid molecule of the invention, host cells into which the expression vectors have been introduced and non-human transgenic animals in which a CARD-3, CARD-4L, CARD-4S, CARD-4Y, CARD-4Z, and murine CARD-4L gene has been introduced or disrupted. The invention further provides CARD-3 and CARD-4 target proteins that bind to CARD-3 or CARD-4 and allelic variants of human CARD-4. Diagnostic, screening and therapeutic methods utilizing compositions of the invention are also provided.

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=>
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=> s (RICK OR RIP2) (4A) (dominant negative)
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  81 FILES SEARCHED...
  95 FILES SEARCHED...
L7      27 (RICK OR RIP2) (4A) (DOMINANT NEGATIVE)

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L9      12 L8 NOT (COMPLETE GENOME)

=> d l9 1-12 bib ab

L9      ANSWER 1 OF 12  BIOSIS  COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN      2002:430172  BIOSIS
DN      PREV200200430172
TI      Involvement of receptor-interacting protein 2 in innate and adaptive
        immune responses.
AU      Chin, Arnold I.; Dempsey, Paul W.; Bruhn, Kevin; Miller, Jeff F.; Xu,
        Yang; Cheng, Genhong [Reprint author]
CS      Molecular Biology Institute, University of California, Los Angeles, CA,
        90095, USA
        genhongc@microbio.ucla.edu
SO      Nature (London), (14 March, 2002) Vol. 416, No. 6877, pp. 190-194. print.
        CODEN: NATUAS. ISSN: 0028-0836.
DT      Article
LA      English
ED      Entered STN: 14 Aug 2002
        Last Updated on STN: 14 Aug 2002
AB      Host defences to microorganisms rely on a coordinated interplay between
        the innate and adaptive responses of immunity. Infection with
        intracellular bacteria triggers an immediate innate response requiring
        macrophages, neutrophils and natural killer cells, whereas subsequent
        activation of an adaptive response through development of T-helper subtype
        1 cells (TH1) proceeds during persistent infection. To understand the
        physiological role of receptor-interacting protein 2 (Rip2), also known as
        RICK and CARDIAK, we generated mice with a targeted disruption of the gene
        coding for Rip2. Here we show that Rip2-deficient mice exhibit a
        profoundly decreased ability to defend against infection by the
        intracellular pathogen Listeria monocytogenes. Rip2-deficient macrophages
        infected with L. monocytogenes or treated with lipopolysaccharide (LPS)
```

have decreased activation of NF-kappaB, whereas **dominant negative Rip2** inhibited NF-kappaB activation mediated by Toll-like receptor 4 and Nod1. In vivo, Rip2-deficient mice were resistant to the lethal effects of LPS-induced endotoxic shock. Furthermore, Rip2 deficiency results in impaired interferon-gamma production in both TH1 and natural killer cells, attributed in part to defective interleukin-12-induced Stat4 activation. Our data reflect requirements for Rip2 in multiple pathways regulating immune and inflammatory responses.

L9 ANSWER 2 OF 12 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
 AN 2001:396222 BIOSIS
 DN PREV200100396222
 TI A prosurvival function for the p75 receptor death domain mediated via the caspase recruitment domain receptor-interacting protein 2.
 AU Khursigara, Gus; Bertin, John; Yano, Hiroko; Moffett, Howell; DiStefano, Peter S.; Chao, Moses V. [Reprint author]
 CS Skirball Institute for Biomolecular Medicine, New York University School of Medicine, 540 First Avenue, New York, NY, 10016, USA
 chao@saturn.med.nyu.edu
 SO Journal of Neuroscience, (August 15, 2001) Vol. 21, No. 16, pp. 5854-5863. print.
 CODEN: JNRSDS. ISSN: 0270-6474.
 DT Article
 LA English
 ED Entered STN: 22 Aug 2001
 Last Updated on STN: 23 Feb 2002
 AB In addition to promoting cell survival, neurotrophins also can elicit apoptosis in restricted cell types. Recent results indicate that nerve growth factor (NGF) can induce Schwann cell death via engagement of the p75 neurotrophin receptor. Here we describe a novel interaction between the p75 receptor and receptor-interacting protein 2, RIP2 (RICK/CARDIAK), that accounts for the ability of neurotrophins to choose between a survival-versus-death pathway. RIP2, an adaptor protein with a serine threonine kinase and a caspase recruitment domain (CARD), is highly expressed in dissociated Schwann cells and displays an endogenous association with p75. RIP2 binds to the death domain of p75 via its CARD domain in an NGF-dependent manner. The introduction of RIP2 into Schwann cells deficient in RIP2 conferred NGF-dependent nuclear transcription factor-kappaB (NF-kappaB) activity and decreased the cell death induced by NGF. Conversely, the expression of a **dominant-negative** version of **RIP2** protein resulted in a loss of NGF-induced NF-kappaB induction and increased NGF-mediated cell death. These results indicate that adaptor proteins like RIP2 can provide a bifunctional switch for cell survival or cell death decisions mediated by the p75 neurotrophin receptor.

L9 ANSWER 3 OF 12 CAPLUS COPYRIGHT 2004 ACS on STN
 AN 2002:616256 CAPLUS
 DN 137:181594
 TI Dominant-negative variants of human protein kinases that inhibit the phosphorylation activity of their active enzyme isoforms
 IN Levine, Zurit; Bernstein, Jeanne
 PA Compugen Ltd., Israel
 SO U.S. Pat. Appl. Publ., 170 pp., Cont.-in-part of U.S. Ser. No. 724,676.
 CODEN: USXXCO
 DT Patent
 LA English
 FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2002110811	A1	20020815	US 2001-771161	20010126
PRAI	IL 2000-135619	A	20000512		
	IL 2000-136776	A	20000615		

US 2000-724676 A2 20001128

AB The present invention concerns 91 nucleic acid sequences and amino acid sequences of variants of various human kinases, i.e. of sequences which inhibit activity of kinases in a dominant manner. The variants lack a domain or region required for phosphorylation, and thus may be dominant-neg. kinases obtained by alternative splicing of known original sequences of the kinase genes. The novel dominant-neg. kinase variants of the invention are not merely artificially truncated forms, fragments or mutations of known genes, but rather novel sequences which naturally occur within the body of individuals. The invention also concerns pharmaceutical compns. and detection methods using these sequences.

L9 ANSWER 4 OF 12 CAPLUS COPYRIGHT 2004 ACS on STN

AN 2002:429126 CAPLUS

DN 137:16563

TI Nod2 nucleic acids and proteins and the association of sequence variants with Crohn's disease

IN Nunez, Gabriel; Inohara, Naohiro; Ogura, Yasunori; Cho, Judy; Nicolae, Dan L.; Bonen, Denise

PA Regents of the University of Michigan, USA; The University of Chicago

SO PCT Int. Appl., 316 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002044426	A2	20020606	WO 2001-US51068	20011026
	WO 2002044426	A3	20040108		
	W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
	RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
	AU 2002043415	A5	20020611	AU 2002-43415	20011026
	US 2002197616	A1	20021226	US 2001-2974	20011026
	EP 1404712	A2	20040407	EP 2001-989310	20011026
	R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR			

PRAI US 2000-244266P P 20001030

US 2001-286316P P 20010425

WO 2001-US51068 W 20011026

AB The present invention relates to intracellular signaling mols., in particular the human Nod2 protein and nucleic acids encoding the Nod2 protein. The Nod2 gene is located on human chromosome 16q12, and shown to comprise 12 coding exons; expression is abundant in monocytes and leukocytes. Activation of NF- κ B by Nod2 requires IKK γ and is inhibited by **dominant neg.** forms of IKK and **RICK**. The present invention provides isolated nucleotide sequence encoding Nod2, isolated Nod2 peptides, antibodies that specifically bind Nod2, methods for the detection of Nod2, and methods for screening compds. for the ability to alter Nod2 associated signal transduction. The present invention also provides Nod2 variant alleles, which are discovered to be associated with the risk of developing inflammatory bowel disease or Crohn's disease. Thus, the present invention further provides methods of identifying individuals at increased risk of developing Crohn's disease.

L9 ANSWER 5 OF 12 DISSABS COPYRIGHT (C) 2004 ProQuest Information and Learning Company; All Rights Reserved on STN

AN 2002:15023 DISSABS Order Number: AAI3020171

TI NGF signaling in Schwann cells: Identification of two p75 interacting proteins
AU Khursigara, Gus [Ph.D.]; Chao, Moses [adviser]
CS Cornell University Medical College (0967)
SO Dissertation Abstracts International, (2001) Vol. 62, No. 7B, p. 3092.
Order No.: AAI3020171. 194 pages.
ISBN: 0-493-32686-3.

DT Dissertation

FS DAI

LA English

AB Neurotrophins were identified because of their ability to promote survival of post mitotic neurons. Since their discovery, the function of neurotrophins has expanded beyond neuronal survival and they have been shown to mediate neuronal differentiation, neurite outgrowth, axon guidance, synaptic plasticity and cell death. These are accomplished by signaling through two receptors, the receptor tyrosine kinase family (Trk) and the p75 neurotrophin receptor.

The activation of the Trk receptor is necessary to mediate many of the tropic effects of neurotrophins including survival and differentiation. The functional role for p75, however, remains unclear. It has been suggested that p75 acts in concert with TrkA to enhance NGF signaling by creating high affinity binding sites and potentiating survival signals. Recently, p75 has been demonstrated to signal autonomously, mediating neurotrophin induced cell death.

The mechanism of p75 induced cell death is not known, but evidence suggests that activation of JNK is involved. In addition, p75 can promote survival independent of Trk signaling, possibly through the activation of NF- κ B. The goal of this thesis was to identify proteins in the molecular pathway of p75 that initiate NF- κ B transcription and JNK activity.

This work identified two p75 interacting proteins, TRAF6 and RIP2. TRAF6 interacts with p75 in a ligand dependent manner, and is necessary to activate NGF-p75 induced ATF-2 activity. A dominant negative TRAF6 expressed in Schwann cells blocked NGF induced cell death. These results suggest that p75 recruits TRAF6 increases JNK activity and to induce cell death in Schwann cells. RIP2 is recruited to the death domain of p75 in a ligand dependent manner. A **dominant negative**

RIP2 inhibited NGF's ability to activate NF- κ B in Schwann cells, and allowed NGF to induce cell death. Taken together these results suggest that RIP2 activates NF- κ B to play a survival role in Schwann cells, whereas TRAF6 activates JNK and can induce cell death. Identification of these p75 adaptor proteins will identify a physiological role for p75 signaling.

L9 ANSWER 6 OF 12 USPATFULL on STN

AN 2003:318644 USPATFULL

TI RIP2: a mediator of signaling in the innate and adaptive immune systems

IN Flavell, Richard A., Guilford, CT, UNITED STATES

Medzhitov, Ruslan M., Branford, CT, UNITED STATES

Kobayashi, Koichi, Branford, CT, UNITED STATES

PI US 2003224388 A1 20031204

AI US 2003-339636 A1 20030109 (10)

PRAI US 2002-348172P 20020109 (60)

DT Utility

FS APPLICATION

LREP ROPES & GRAY LLP, ONE INTERNATIONAL PLACE, BOSTON, MA, 02110-2624

CLMN Number of Claims: 52

ECL Exemplary Claim: 1

DRWN 6 Drawing Page(s)

LN.CNT 1217

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB This invention provides a method of identifying a compound that modulates an innate immune response and an adaptive immune response comprising contacting cells expressing RIP2 with a candidate compound,



130252.0

#7

PATENT
09/771,161
802620-2005.1
130252.0**DECLARATION FOR PATENT APPLICATION**

As below named inventors, we, Zurit Levine and Jeanne Bernstein, hereby declare:

Our residences and citizenships are below. We believe we are original, first and joint inventors of the subject matter claimed on the invention entitled

VARIANTS OF PROTEIN KINASES

for which an application for Letters Patent was filed on January 26, 2001 and accorded United States Application No. 09/771,161.

We have reviewed and understand the contents of the above-identified specification and the claims. We acknowledge the duty to disclose to the United States Patent and Trademark Office (US PTO) all information material to patentability known to us as defined in 37 CFR § 1.56.

We claim foreign priority benefits under 35 USC § 119 of Israel Patent applications 136776 and 135619 filed 15 June 2000 and 12 April 2000, respectively.

We claim the benefit under 35 USC § 120 of the United States application Serial No. 09/724,676 filed 28 November 2000 and, insofar as the subject matter of each of the claims of this application is not disclosed in that prior application in the manner provided by the first paragraph of 35 USC § 112, we acknowledge the duty to disclose to the US PTO all information known to us to be material to patentability as defined in 37 CFR § 1.56 which became available between the filing date of the prior application and the filing date of this application.

We declare that all statements made herein of our own knowledge are true and that all statements made on information and belief are believed to be true; and further that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under USC 18 § 1001 and that such willful false statements may jeopardize the validity of the application or any patent issued thereon.

Signature: X April 11, 2001
Full name of first inventor: **ZURIT LEVINE**
Residence: Hakozrim Street 5
Herzliya 46360, Israel
Citizenship: Israel

Date: X 2

Signature: X April 9, 2001
Full name of second inventor: **JEANNE BERNSTEIN**
Residence: Harimon Street 23
Kfar Yona 40300, Israel
Citizenship: Israel

Date: X [Signature]

and determining whether the candidate compound modulates RIP2 activity in the cells, wherein modulation of RIP2 activity in the cells by the candidate compound indicates that the candidate compound modulates the innate immune response and adaptive immune response.

L9 ANSWER (7) OF 12 USPATFULL on STN
AN 2003:250976 USPATFULL
TI Modulators on Nod2 signaling
IN Nunez, Gabriel, Ann Arbor, MI, UNITED STATES
Inohara, Naohiro, Ann Arbor, MI, UNITED STATES
Ogura, Yasunori, Ann Arbor, MI, UNITED STATES
PA The Regents of the University of Michigan, Ann Arbor, MI (U.S. corporation)
PI US 2003175762 A1 20030918
AI US 2002-314506 A1 20021209 (10)
RLI Continuation-in-part of Ser. No. US 2001-14269, filed on 26 Oct 2001, PENDING
PRAI US 2000-244289P 20001030 (60)
DT Utility
FS APPLICATION
LREP MEDLEN & CARROLL, LLP, Suite 350, 101 Howard Street, San Francisco, CA, 94105
CLMN Number of Claims: 29
ECL Exemplary Claim: 1
DRWN 33 Drawing Page(s)
LN.CNT 4803
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to intracellular signaling molecules, in particular the Nod2 protein and nucleic acids encoding the Nod2 protein. The present invention provides methods of identifying modulators of Nod2 signaling. In particular, the present invention additionally provides methods of screening immune modulators such as adjuvants using Nod2. The present invention further provides methods of altering Nod2 signaling.

L9 ANSWER (8) OF 12 USPATFULL on STN
AN 2002:343967 USPATFULL
TI Novel molecules of the card-related protein family and uses thereof
IN Bertin, John, Watertown, MA, UNITED STATES
PA Millennium Pharmaceuticals, Inc., a Delaware corporation (U.S. corporation)
PI US 2002197693 A1 20021226
AI US 2002-118984 A1 20020409 (10)
RLI Division of Ser. No. US 1999-245281, filed on 5 Feb 1999, GRANTED, Pat. No. US 6369196 Continuation-in-part of Ser. No. US 1998-207359, filed on 8 Dec 1998, GRANTED, Pat. No. US 6469140 Continuation-in-part of Ser. No. US 1998-99041, filed on 17 Jun 1998, GRANTED, Pat. No. US 6340576 Continuation-in-part of Ser. No. US 1998-19942, filed on 6 Feb 1998, GRANTED, Pat. No. US 6033855
DT Utility
FS APPLICATION
LREP ANITA L. MEIKLEJOHN, PH.D., Fish & Richardson P.C., 225 Franklin Street, Boston, MA, 02110-2804
CLMN Number of Claims: 22
ECL Exemplary Claim: 1
DRWN 36 Drawing Page(s)
LN.CNT 4142
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel CARD-3, CARD-4L, CARD-4S, CARD-4Y, CARD-4Z, and murine CARD-4L polypeptides, proteins, and nucleic acid molecules are disclosed. In addition to isolated CARD-3, CARD-4L, CARD-4S, CARD-4Y, CARD-4Z, and murine CARD-4L proteins, and the invention further provides CARD-3, CARD-4L, CARD-4S, CARD-4Y, CARD-4Z, and murine CARD-4L fusion proteins, antigenic peptides and anti-CARD-3, anti-CARD-4L and anti-CARD-4S, anti-CARD-4Y, anti-CARD-4Z, and anti-murine CARD-4L antibodies. The

invention also provides CARD-3, CARD-4L, CARD-4S, CARD-4Y, CARD-4Z, and murine CARD-4L nucleic acid molecules, recombinant expression vectors containing a nucleic acid molecule of the invention, host cells into which the expression vectors have been introduced and non-human transgenic animals in which a CARD-3, CARD-4L, CARD-4S, CARD-4Y, CARD-4Z, and murine CARD-4L gene has been introduced or disrupted. The invention further provides CARD-3 and CARD-4 target proteins that bind to CARD-3 or CARD-4 and allelic variants of human CARD-4. Diagnostic, screening and therapeutic methods utilizing compositions of the invention are also provided.

L9 ANSWER 9 OF 12 USPATFULL on STN
AN 2002:343890 USPATFULL
TI Nod2 nucleic acids and proteins
IN Nunez, Gabriel, Ann Arbor, MI, UNITED STATES
Inohara, Naohiro, Ann Arbor, MI, UNITED STATES
Ogura, Yasunori, Ann Arbor, MI, UNITED STATES
Cho, Judy, Chicago, IL, UNITED STATES
Nicolae, Dan L., Chicago, IL, UNITED STATES
Bonen, Denise, Chicago, IL, UNITED STATES
PI US 2002197616 A1 20021226
AI US 2001-2974 A1 20011026 (10)
PRAI US 2000-244266P 20001030 (60)
US 2001-286316P 20010425 (60)
DT Utility
FS APPLICATION
LREP David A. Casimir, MEDLEN & CARROLL, LLP, Suite 350, 101 Howard Street,
San Francisco, CA, 94105
CLMN Number of Claims: 33
ECL Exemplary Claim: 1
DRWN 49 Drawing Page(s)
LN.CNT 8372
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to intracellular signalling molecules, in particular the Nod2 protein and nucleic acids encoding the Nod2 protein. The present invention provides isolated nucleotide sequence encoding Nod2, isolated Nod2 peptides, antibodies that specifically bind Nod2, methods for the detection of Nod2, and methods for screening compounds for the ability to alter Nod2 associated signal transduction. The present invention also provides Nod2 variant alleles. The present invention further provides methods of identifying individuals at increased risk of developing Crohn's disease.

L9 ANSWER 10 OF 12 USPATFULL on STN
AN 2002:272872 USPATFULL
TI Novel tumor necrosis factor receptor homolog and nucleic acids encoding the same
IN Ashkenazi, Avi J., San Mateo, CA, UNITED STATES
Goddard, Audrey, San Francisco, CA, UNITED STATES
Gurney, Austin, Belmont, CA, UNITED STATES
Marsters, Scot A., San Carlos, CA, UNITED STATES
Pitti, Robert M., El Cerrito, CA, UNITED STATES
Wood, William I., Hillsborough, CA, UNITED STATES
PA Genentech, Inc. (U.S. corporation)
PI US 2002150993 A1 20021017
AI US 2002-116378 A1 20020404 (10)
RLI Continuation of Ser. No. US 1999-247225, filed on 9 Feb 1999, PENDING
PRAI US 1998-74087P 19980209 (60)
DT Utility
FS APPLICATION
LREP GENENTECH, INC., 1 DNA WAY, SOUTH SAN FRANCISCO, CA, 94080
CLMN Number of Claims: 32
ECL Exemplary Claim: 1
DRWN 15 Drawing Page(s)

L Number	Hits	Search Text	DB	Time stamp
1	29	(human or sapiens) near4 (serine adj kinase)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/04/14 13:19
2	381	(human or sapiens) near4 (serine near3 kinase)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/04/14 13:20
3	3	rick near4 (splice or splicing or variant)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/04/14 13:44
4	0	rip2 near4 (splice or splicing or variant)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/04/14 13:26
5	0	((human or sapiens) near4 (serine near3 kinase)) and (rick near4 (splice or splicing or variant))	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/04/14 13:26
6	7	(rick or rip2) near4 (dominant adj negative)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/04/14 15:45
7	2	"2003250976"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/04/14 15:45
8	19	"250976"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/04/14 15:46



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☐ 1: Chen CM, Gong Y, Zhang M, Chen JJ. Related Articles, Link:

Reciprocal cross talk between Nod2 and TAK1 signaling pathways.
J Biol Chem. 2004 Apr 9 [Epub ahead of print]
PMID: 15075345 [PubMed - as supplied by publisher]

☐ 2: Zhang WH, Wang X, Narayanan M, Zhang Y, Huo C, Reed JC, Friedlander RM. Related Articles, Link:

Fundamental role of the Rip2/caspase-1 pathway in hypoxia and ischemia-induced neuronal cell death.
Proc Natl Acad Sci U S A. 2003 Dec 23;100(26):16012-7. Epub 2003 Dec 08.
PMID: 14663141 [PubMed - in process]

☐ 3: Shikama Y, Yamada M, Miyashita T. Related Articles, Link:

Caspase-8 and caspase-10 activate NF-kappaB through RIP, NIK and IKKalpha kinases.
Eur J Immunol. 2003 Jul;33(7):1998-2006.
PMID: 12884866 [PubMed - indexed for MEDLINE]

☐ 4: Muto A, Ruland J, McAllister-Lucas LM, Lucas PC, Yamaoka S, Chen FF, Lin A, Mak TW, Nunez G, Inohara N. Related Articles, Link:

Protein kinase C-associated kinase (PKK) mediates Bcl10-independent NF-kappa B activation induced by phorbol ester.
J Biol Chem. 2002 Aug 30;277(35):31871-6. Epub 2002 Jun 28.
PMID: 12091384 [PubMed - indexed for MEDLINE]

☐ 5: Chin AI, Dempsey PW, Bruhn K, Miller JF, Xu Y, Cheng G. Related Articles, Link:

Involvement of receptor-interacting protein 2 in innate and adaptive immune responses.
Nature. 2002 Mar 14;416(6877):190-4.
PMID: 11894097 [PubMed - indexed for MEDLINE]


☐ 6: Druilhe A, Srinivasula SM, Razmara M, Ahmad M, Alnemri ES. Related Articles, Link:

Regulation of IL-1beta generation by Pseudo-ICE and ICEBERG, two dominant negative caspase recruitment domain proteins.
Cell Death Differ. 2001 Jun;8(6):649-57.
PMID: 11536016 [PubMed - indexed for MEDLINE]

☐ 7: Khursigara G, Bertin J, Yano H, Moffett H, DiStefano PS, Chao MV. Related Articles, Link:


A prosurvival function for the p75 receptor death domain mediated via the caspase recruitment domain receptor-interacting protein 2.
J Neurosci. 2001 Aug 15;21(16):5854-63.
PMID: 11487608 [PubMed - indexed for MEDLINE]

- ☐ **8:** [Girardin SE, Tournebise R, Mavris M, Page AL, Li X, Stark GR, Bertin J, DiStefano PS, Yaniv M, Sansonetti PJ, Philpott DJ.](#) [Related Articles, Link](#)

 **CARD4/Nod1 mediates NF-kappaB and JNK activation by invasive Shigella flexneri.**


EMBO Rep. 2001 Aug;2(8):736-42. Epub 2001 Jul 19.
PMID: 11463746 [PubMed - indexed for MEDLINE]

- ☐ **9:** [Ogura Y, Inohara N, Benito A, Chen FF, Yamaoka S, Nunez G.](#) [Related Articles, Link](#)

 **Nod2, a Nod1/Apaf-1 family member that is restricted to monocytes and activates NF-kappaB.**


J Biol Chem. 2001 Feb 16;276(7):4812-8. Epub 2000 Nov 21.
PMID: 11087742 [PubMed - indexed for MEDLINE]

- ☐ **10:** [Yu PW, Huang BC, Shen M, Quast J, Chan E, Xu X, Nolan GP, Payan DG, Luo Y.](#) [Related Articles, Link](#)

 **Identification of RIP3, a RIP-like kinase that activates apoptosis and NFkappaB.**

Curr Biol. 1999 May 20;9(10):539-42.
PMID: 10339433 [PubMed - indexed for MEDLINE]

- ☐ **11:** [Koseki T, Inohara N, Chen S, Carrio R, Merino J, Hottiger MO, Nabel GJ, Nunez G.](#) [Related Articles, Link](#)

 **CIPER, a novel NF kappaB-activating protein containing a caspase recruitment domain with homology to Herpesvirus-2 protein E10.**

J Biol Chem. 1999 Apr 9;274(15):9955-61.
PMID: 10187770 [PubMed - indexed for MEDLINE]

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Caspase-1 activates NF-kappa B independent of its enzymatic activity.
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